Household Income and Cardiovascular Disease Risks in U.S. Children and Young Adults

Analyses from NHANES 1999–2008

Mohammed K. Ali, mbchb, msc[1,2]
Kai McKeever Bullard, mph, phd[2]
Gloria L. Beckles, md, msc[2]
Mark R. Stevens, msph, ma[3]
Lawrence Barker, phd[2]
K.M. Venkat Narayan, md[1]
Giuseppina Imperatore, md, phd[2]

OBJECTIVE—To assess the cardiovascular risk profile of youths across socioeconomic groups in the U.S.

RESEARCH DESIGN AND METHODS—Analysis of 1999–2008 National Health and Nutrition Examination Surveys (NHANES) including 16,085 nonpregnant 6- to 24-year-olds to estimate race/ethnicity-adjusted prevalence of obesity, central obesity, sedentary behaviors, tobacco exposure, elevated systolic blood pressure, glycated hemoglobin, non-HDL cholesterol (non–HDL-C), and high-sensitivity C-reactive protein according to age-group, sex, and poverty-income ratio (PIR) tertiles.

RESULTS—Among boys aged 6–11 years, 19.9% in the lowest PIR tertile were obese and 30.0% were centrally obese compared with 13.2 and 21.6%, respectively, in the highest-income tertile households ($P_{obesity} < 0.05$ and $P_{central\ obesity} < 0.01$). Boys aged 12–17 years in lowest-income households were more likely than their wealthiest family peers to be obese (20.6 vs. 15.6%, $P < 0.05$), sedentary (14.8 vs. 9.3%, $P < 0.05$), and exposed to tobacco (19.0 vs. 6.5%, $P < 0.01$). Compared with girls aged 12–17 years in highest-income households, lowest-income household girls had higher prevalence of obesity (17.9 vs. 13.1%, $P < 0.05$), central obesity (41.5 vs. 29.2%, $P < 0.01$), sedentary behaviors (20.4 vs. 9.4%, $P < 0.01$), and tobacco exposure (14.1 vs. 5.9%, $P < 0.01$). Apart from higher prevalence of elevated non–HDL-C among low-income women aged 18–24 years (23.4 vs. 15.8%, $P < 0.05$), no other cardiovascular disease risk factor prevalence differences were observed between lowest- and highest-income background young adults.

CONCLUSIONS—Independent of race/ethnicity, 6- to 17-year-olds from low-income families have higher prevalence of obesity, central obesity, sedentary behavior, and tobacco exposure. Multifaceted cardiovascular health promotion policies are needed to reduce health disparities between income groups.

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Socioeconomic position (SEP) is a multidimensional construct, embodying prestige, relationships, opportunities, and access to resources (1,2). In adults, low SEP, measured as lower education level, self-reported economic difficulties, or impoverished locality, strongly predicts increased cardiovascular disease (CVD) risk factors, CVD events, and mortality (3). Increased adult CVD risk is also related to adverse early life circumstances, independent of adult SEP (2). This relationship may be mediated by early onset of risk factors that track into adulthood and accelerate progression of atherosclerosis (4,5). However, evidence of the relationship between childhood SEP and CVD risks in early life is limited, inconsistent, and suggests variation by sex and age (6,7).

In the U.S., CVD risk factors are increasingly common in youth, exhibit racial/ethnic variation, and have been attributed to a myriad of interconnected factors (genetic influences, birth weight, parental BMI, nutrient quantity and quality, and activity levels) (8,9). Among U.S. children and adolescents, socioeconomic disadvantage is common, varies by race/ethnicity, and persists into young adulthood (10). Therefore, examining the contribution of SEP to emergence of early cardiometabolic risk may provide insights into origins of socioeconomic disparities in CVD risk and has implications for societal action and policies aimed at preventing and controlling health and economic burdens across the life course. Furthermore, identifying and evaluating specific, modifiable exposure-outcome associations and the strength of these relationships may help guide the design of appropriate interventions.

We used recent nationally representative data to determine whether SEP is associated with prevalence of common CVD risk factors in U.S. youth, independent of related sociodemographic covariates (race/ethnicity, age, and sex).

RESEARCH DESIGN AND METHODS

—We analyzed data from National Health and Nutrition Examination Surveys (NHANES) 1999–2008, collected by the National Center for Health Statistics at the Centers for Disease Control and Prevention. This cross-sectional survey uses stratified multistage probability cluster sampling to ensure adequate representation of the nation's noninstitutionalized civilian population. Trained interviewers conduct household interviews, collecting demographic, SEP, and medical history data. Participants are invited to attend mobile examination centers for

From the [1]Rollins School of Public Health, Emory University, Atlanta, Georgia; the [2]Division of Diabetes Translation, Centers for Disease Control and Prevention, Atlanta, Georgia; and the [3]National Centers for Injury Prevention and Control, Centers for Disease Control and Prevention, Atlanta, Georgia.
Corresponding author: Mohammed K. Ali, mkali@emory.edu.
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additional questionnaires, standardized medical examinations (11) (including anthropometric measurements), and blood sample collection.

The study received approval for human subjects research from the Centers for Disease Control and Prevention. Survey instruments and protocols have been described extensively (11). Overall survey response rates were 76% (1999–2000), 80% (2001–2002), 76% (2003–2004), 77.4% (2005–2006), and 75.4% (2007–2008).

The analytic sample included 16,085 participants aged 6–24 years, after excluding participants who did not complete the examination (n = 660); those of race/ethnicity other than non-Hispanic white (NHW), non-Hispanic black (NHB), or Mexican American (MA) (n = 1,652); those with diagnosed diabetes (n = 76); and pregnant women (n = 545).

Outcome variables

Measurement and definitions of CVD risk factors (11,12) follow. Specifically, obesity was defined as BMI (kg/m^2) greater than or equal to age- and sex-specific 95th percentile for youth aged 6–17 years (13) and BMI ≥30 kg/m^2 for young adults aged 18–24 years.

Central obesity was defined as a waist-to-height ratio ≥0.5 for all participants (14).

Sedentary behavior was defined as reporting no physical activity during past month. Participants aged ≥12 years were asked to report frequency and duration of leisure-time physical activity in the past month. Physical activity was coded and classified according to Ainsworth's standardized scheme (15). Because of modification in physical activity assessments in 2007, analyses assessing sedentary behavior were limited to 1999–2006 data.

Tobacco exposure (active and/or passive) was defined as serum cotinine levels >73.84 nmol/L (13 ng/mL) (16). Cotinine was measured using internal diameter high-performance liquid chromatography–atmospheric-pressure chemical ionization mass spectrometry methods in all participants.

Elevated systolic blood pressure (sBP; mmHg) was defined as ≥90th sex-, age-, and height-specific percentile (8- to 17-year-olds) or sBP ≥140 mmHg (≥18 years). The average of three brachial artery BP readings (taken by physicians after 5 min in a rested sitting position) (11) was used for analysis.

Elevated glycated hemoglobin (HbA_{1c}) was defined as ≥0.057 (5.7%). HbA_{1c} was measured in participants aged ≥12 years from whole blood using Diamat HPLC (high-performance liquid chromatography; BioRad Laboratories, Columbia, MO [1999–2006]) and A_{1c} G7 HPLC Glycohemoglobin Analyzer (Tosoh Medics, Inc., San Francisco, CA [2007–2008]). Assays were standardized to reference methods from the Diabetes Control and Complications Trial (12).

Elevated non-HDL cholesterol (non–HDL-C) was calculated as total cholesterol minus HDL-C. Levels ≥3.73 mmol/L (144 mg/dL) equivalent to adult LDL ≥3.37 mmol/L (130 mg/dL) were considered elevated (17). In all participants, total cholesterol and HDL-C were measured using Roche/Boehringer-Mannheim Diagnostics methods.

Elevated high sensitivity C-reactive protein (hs-CRP) was defined as ≥10.0 mg/L (1.0 mg/dL), which corresponds to 75th percentile in published data and intermediate CVD risk in adults (18). Individuals with markedly elevated hs-CRP (≥150.0 mg/L [15.0 mg/dL]) were excluded (presumed to be experiencing acute phase responses of inflammatory conditions). Serum hs-CRP was estimated in all participants using latex-enhanced nephelometry.

Among participants aged 8–24 years, we calculated the proportion with 1, 2, or ≥3 of the following risk factors: central obesity, elevated sBP, elevated hs-CRP, high non–HDL-C, and tobacco exposure. Data for HbA_{1c} and sedentary behavior were not available for children <12 years.

Principal exposure variable

Household income is a dynamic, potentially modifiable indicator of SEP, reflecting current material resources (2). We used poverty-income ratio (PIR), an index of income in relation to family need, derived from household income and federally established poverty thresholds (based on family size and annual changes in cost of living [tracking Consumer Price Index]) (19). We categorized PIR into tertiles (lowest, T1 [PIR: 0–1.37]; medium, T2 [1.38–3.25]; and highest, T3 [≥3.26]). A small fraction of 18- to 24-year-olds (5.7%) reported living alone; however, distribution across SEP strata reflected national levels.

Covariates

Participants self-identified as NHW, NHB, or MA. Participants' age was categorized into childhood (6–11 years), adolescence (12–17 years), and young adulthood (18–24 years).

Data analysis

Data were analyzed using SAS version 9.2 (SAS Institute Inc., Cary, NC) and SUDAAN version 10.0 (Research Triangle Institute, Research Triangle Park, NC) with adjustment for NHANES' complex survey design. We used multiple imputation to account for missing income data (n = 1,235). Income was selected for imputation because it had the highest rate of missing cases; missing data on CVD risk factors (range: 0–16% missing; average: 7.3% missing per outcome) were not imputed since these outcomes are less predictable. Missing income and PIR were predicted based on race/ethnicity, household size, and household reference person's age and sex. Ten imputed datasets were created and imputations were combined. In addition, we performed sensitivity analyses isolating participants with missing PIR data in a separate category to identify whether CVD risk profiles in this group deviated markedly from participants without missing data.

Following descriptive explorations of population characteristics, sex-specific logistic regression models controlling for covariates were used to investigate the main effects of PIR on CVD risk. We included all first-order interactions and tested their significance using design-adjusted Wald F tests to examine whether SEP-CVD relationship(s) varied by other independent variables (age and race/ethnicity).

To estimate the proportion of participants with each CVD risk factor in each PIR tertile, sex- and age-specific predicted marginal probabilities (a type of standardization in which predicted values from regression models are averaged over the entire sample's covariate distribution) and 95% CIs were estimated. We tested whether risk factor differences between PIR groups were significant using Wald F tests.

We also used multinomial logistic regression to calculate the proportion of participants with 1, 2, or ≥3 risk factors by PIR tertile and tested for between-tertile differences and age-PIR interaction in youth with ≥3 risk factors.

All tests were two-sided. Results were considered significant if $P < 0.05$.

RESULTS—Table 1 presents the sociodemographic characteristics of the study population. Of note, 21.4% of children and adolescents (aged 6–17 years) lived in households with income below poverty level (PIR <1). Among young adults (aged 18–24 years), this proportion was

Table 1—*Sociodemographic profile of U.S. NHWs, NHBs, and MAs aged 6–24 years, NHANES 1999–2008* (N = *16,085*)

	n	Population size*	Weighted %
Age (%)			
6–11 Years	5,027	21.3	32.0
12–17 Years	7,153	21.9	33.0
18–24 Years	3,905	23.3	35.0
Mean age (SE)			14.9 (0.1)
Sex (%)			
Female	7,881	32.0	48.2
Male	8,204	34.5	51.8
Ethnicity/Race (%)			
NHW	5,021	46.4	69.8
NHB	5,435	11.0	16.5
MA	5,629	9.1	13.7
Household size (%)			
≤2	1,368	8.3	12.5
3 to 5	10,368	46.2	69.5
≥6	4,349	12.0	18.0
Mean household size (SE)			4.2 (0.03)
Household income (%)†			
≤$24,999	6,964	23.5	35.3
$25,000–49,999	3,463	12.8	19.3
$50,000–74,999	2,882	13.3	20.0
≥$75,000	2,776	16.9	25.4
Poverty level (%)†			
6–17 Years			
Below poverty level (PIR <1)	3,850	9.1	21.1
At or above poverty level (PIR ≥1)	8,330	34.1	78.9
18–24 Years			
Below poverty level (PIR <1)	1,336	6.2	26.5
At or above poverty level (PIR ≥1)	2,569	17.1	73.5

*In millions. †Missing values for income group (n = 972) and poverty level (n = 1,235) were estimated using multiple imputation.

26.5%. Those with missing PIR data (subsequently imputed) were more likely to be 18 years or older, more educated, NHB or MA, and living in larger households.

Accounting for age and race/ethnicity (Table 2) and relative to highest PIR tertile, lower PIR was significantly associated with greater odds of sedentary behavior (P_{males} = 0.02, $P_{females}$ = 0.001) and tobacco exposure (P_{males} = 0.003, $P_{females}$ = 0.003) in both sexes. In addition, among females, lower PIR was associated with central obesity (P = 0.001) and elevated non–HDL-C (P = 0.007). In fully adjusted models with main effects and all first-order interactions (results not shown), significant PIR × age interactions were detected in males for central obesity (P = 0.03) and in both sexes for tobacco exposure ($P_{males} < 0.001$, $P_{females} < 0.001$).

Table 3 presents the sex-specific marginal predicted prevalence of anthropometric, behavioral, and metabolic CVD risk factors among youth by PIR tertile. We noted statistically significant differences in risk factor prevalence between highest and lowest PIR groups. Lowest PIR tertile boys aged 6–11 years had a higher prevalence of obesity (T1: 19.9% [95% CI 16.8–23.0] vs. T3: 13.2% [9.5–16.9]) and central obesity (T1: 30.0% [25.7–34.3] vs. T3: 21.6% [17.5–25.7]) compared with their wealthiest counterparts. Adolescent boys aged 12–17 years in lowest SEP groups were more obese (T1: 20.6% [17.9–23.3] vs. T3: 15.6% [12.5–18.7]) and more likely to be sedentary (T1: 14.8% [11.5–18.1] vs. T3: 9.3% [6.2–12.4]) and exposed to tobacco (T1: 19.0% [14.9–23.1] vs. T3: 6.5% [4.7–8.3]). Across all age-groups in boys and young men, there were no significant differences between highest and lowest SEP in terms of elevated sBP, non–HDL-C, HbA_{1c}, or hs-CRP.

Among girls aged 6–11 years, the lowest SEP group had higher prevalence of elevated hs-CRP (T1: 3.5% [95% CI 1.9–5.1] vs. T3: 0.6% [−0.2 to 1.4]); a similar pattern was noted for adolescent girls aged 12–17 years (T1: 4.2% [2.6–5.8] vs. T3: 1.7% [0.5–2.9]). A greater proportion of lowest SEP girls aged 12–17 years were classified as obese (T1: 17.9% [14.0–21.8] vs. T3: 13.1% [10.4–15.8]) and centrally obese (T1: 41.5% [36.6–46.4] vs. T3: 29.2% [25.7–32.7]) compared with highest SEP girls. Twice the proportion of adolescent girls from lowest-income households were sedentary (T1: 20.4% [16.9–23.9] vs. T3: 9.4% [6.5–12.3]) and exposed to tobacco (T1: 14.1% [10.4–17.8] vs. T3: 5.9% [3.9–7.9]) than adolescent girls from highest-income households. Young adult women in the poorest PIR tertile exhibited higher prevalence of elevated non–HDL-C (T1: 23.4% [18.7–28.1] vs. T3: 15.8% [11.5–20.1]) compared with their wealthiest peers. In girls and women of all ages, there were no differences in the prevalence of elevated sBP or HbA_{1c} between PIR groups.

Across both sexes and all age-groups, 19.4–42.5% had one risk factor (Fig. 1). The proportion of youth with 2 or ≥3 CVD risk factors was greater in older age categories across all PIR groups. We noted statistically significant income-group differences in risk factor clustering (≥3 risks) among boys aged 8–11 years (T1: 5.0% [95% CI 0.7–9.3] vs. T3: 0.7% [−0.7 to 2.1]), girls aged 8–11 years (T1: 3.1% [0.7–5.5] vs. T3: 0.7% [−0.5 to 1.9]), and girls aged 12–17 years (T1: 2.6% [1.0–4.2] vs. T3: 1.0% [0.2–1.8]). We found no significant age-PIR interactions for the presence of multiple risk factors (P_{males} = 0.17, $P_{females}$ = 0.49).

CONCLUSIONS—This study shows that independent of race/ethnicity, economically disadvantaged youth had a worse CVD risk profile. The pattern of SEP-CVD risk factor associations was more noteworthy and consistent for proximal behavioral (sedentary behavior and tobacco exposure) than for metabolic risk factors (elevated non–HDL-C, HbA_{1c}, sBP, and hs-CRP). This study's findings add to existing literature that shows household socioeconomic circumstances (20) and access (to material, information, social, and environmental resources) affect health outcomes through shaping health-related awareness and behaviors (dietary preferences, activity levels, and tobacco use) (21).

In our data, socioeconomic disparities in behavioral-environmental risk factors

(physical inactivity and tobacco exposure) were similar in both sexes and more strongly evident among adolescents but not young adults. This may be related to similarly high prevalence of behavioral and anthropometric risks across all income tertiles in young adults. For example, we observed large differences between SEP strata in the prevalence of central obesity in boys and tobacco exposure in both sexes, among the youngest age-groups, which tended to diminish with age. This highlights how age-related transitions and social context adjustments (e.g., adoption of detrimental health behaviors in response to different psychosocial, environmental [school, college, and early career], or socioeconomic stressors) may contribute to behavioral and anthropometric changes.

Our data further highlight that the pathways mediating SEP influences on risk factor emergence are nonlinear and complex (22). The sociodemographic and economic exposures that cumulatively define SEP are interconnected (1,2,22). In particular, socioeconomic disparities in the U.S. mirror race/ethnicity differences with worse CVD risk profiles noted among minority race/ethnicity youth (8,23). In analyses accounting for PIR (Table 2), we found NHB and MA youth exhibited significantly higher odds of obesity, central obesity, sedentary behavior, elevated sBP, elevated non–HDL-C, and elevated HbA_{1c} but also lower likelihood of tobacco exposure—these patterns were broadly consistent with 1988–1994 NHANES estimates (8). Also, in keeping with previous estimates, lowest PIR group youth of all races/ethnicities were significantly more likely to be sedentary and exposed to tobacco. These findings identify specific behavioral risks among youth that can be targeted with health promotion strategies and highlight that poverty is associated with health risks among all race/ethnic groups.

These analyses are, however, cross-sectional. Thus, temporal and causal inferences are limited, as are estimations of population-level cardiovascular benefits from alleviating poverty in lowest PIR tertiles. We transparently used sophisticated imputation and sensitivity analyses to address limitations of missing income data; sensitivity analyses treating missing PIR data as a separate category showed CVD risk factor prevalence estimates in this group were no different from those in referent highest PIR tertile. Furthermore, our NHANES-derived estimates of

Table 2—*Adjusted odds ratios (95% CIs)* for CVD risk factors among U.S. NHWs, NHBs, and MAs aged 6–24 years*

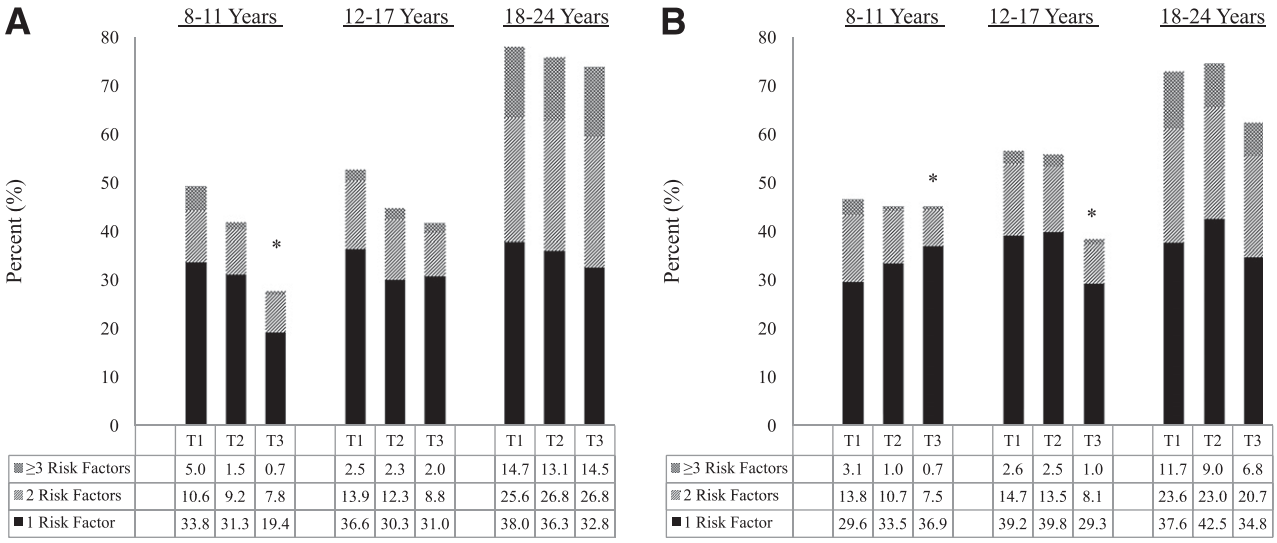
	Obesity	Central obesity	Sedentary behavior	Tobacco exposure	High sBP†	High non–HDL-C	High HbA_{1c}	High hs-CRP
Males								
PIR (tertiles)								
1	1.2 (1.0–1.5)	1.2 (1.0–1.4)	1.6 (1.1–2.2)	1.7 (1.2–2.3)	1.2 (0.8–1.7)	0.9 (0.7–1.1)	1.3 (0.7–2.3)	1.5 (0.9–2.5)
2	1.2 (0.9–1.5)	1.2 (1.0–1.4)	1.2 (0.8–1.7)	1.3 (1.0–1.7)	1.0 (0.7–1.5)	0.8 (0.7–1.1)	1.0 (0.6–1.7)	1.4 (0.8–2.3)
3 (referent)								
Race/Ethnicity								
NHW (referent)								
NHB	1.2 (1.0–1.4)	0.6 (0.5–0.7)	1.4 (1.1–1.9)	0.7 (0.5–0.9)	1.4 (1.0–1.8)	0.8 (0.6–1.0)	9.1 (5.1–16.1)	1.1 (0.7–1.7)
MA	1.4 (1.2–1.7)	1.8 (1.4–2.1)	2.2 (1.7–2.7)	0.3 (0.2–0.4)	1.0 (0.8–1.3)	1.0 (0.9–1.2)	2.7 (1.4–5.3)	1.1 (0.7–1.8)
Age (years)								
6–11	0.9 (0.8–1.1)	0.9 (0.8–1.0)		N/A‡	1.2 (0.9–1.6)	1.0 (0.8–1.2)		1.1 (0.7–1.8)
12–17 (referent)								
18–24	1.1 (0.9–1.4)	2.0 (1.7–2.4)	2.1 (1.7–2.7)	7.0 (5.9–8.4)	2.3 (1.8–2.9)	2.4 (1.8–3.1)	0.8 (0.5–1.1)	1.6 (1.1–2.5)
Females								
PIR (tertiles)								
1	1.4 (1.0–1.8)	1.4 (1.1–1.8)	1.9 (1.3–2.8)	2.0 (1.3–2.9)	1.0 (0.6–1.5)	1.5 (1.2–2.0)	1.0 (0.5–2.1)	1.5 (0.8–2.9)
2	1.3 (1.0–1.8)	1.5 (1.2–1.8)	1.4 (1.0–2.1)	1.5 (1.1–2.2)	1.2 (0.7–1.8)	1.4 (1.0–1.8)	0.8 (0.4–1.9)	1.4 (0.8–2.6)
3 (referent)								
Race/Ethnicity								
NHW (referent)								
NHB	2.0 (1.7–2.4)	1.1 (0.9–1.3)	1.9 (1.5–2.5)	0.5 (0.4–0.7)	1.6 (1.1–2.4)	0.7 (0.6–0.9)	5.1 (2.6–10.1)	1.4 (1.0–2.0)
MA	1.4 (1.1–1.7)	1.8 (1.5–2.2)	1.9 (1.4–2.6)	0.2 (0.1–0.3)	1.0 (0.7–1.5)	0.7 (0.5–0.8)	1.7 (0.9–3.4)	1.4 (1.0–2.0)
Age (years)								
6–11	1.0 (0.8–1.2)	0.7 (0.6–0.9)		N/A‡	1.4 (1.0–2.0)	1.4 (1.0–1.9)		0.8 (0.4–1.4)
12–17 (referent)								
18–24	1.6 (1.3–2.0)	1.9 (1.6–2.4)	1.6 (1.3–2.0)	3.5 (2.6–4.6)	0.5 (0.3–0.8)	2.2 (1.8–2.8)	1.14 (0.7–1.9)	2.7 (1.8–4.1)

*Logistic regression models of CVD risk factors controlling for PIR, race/ethnicity, and age. †Measured in individuals aged ≥8 years. ‡Cases of cotinine >13 ng/mL among 6- to 11-year-olds too low to permit analyses.

Table 3—*Predicted marginal CVD risk factor prevalence among U.S. NHWs, NHBs, and MAs aged 6–24 years by PIR tertile and age-group*

	6–11 Years			12–17 Years			18–24 Years			
	T1	T2	T3	T1	T2	T3	T1	T2	T3	*P* value†
Males										
Obesity	19.9 (16.8–23.0)	18.6 (14.5–22.7)	13.2‡ (9.5–16.9)	20.6 (17.9–23.3)	18.9 (15.6–22.2)	15.6‡ (12.5–18.7)	17.9 (14.4–21.4)	20.4 (15.9–24.9)	22.0 (16.5–27.5)	0.08
Central obesity	30.0 (25.7–34.3)	28.9 (24.6–33.2)	21.6§ (17.5–25.7)	31.9 (28.4–35.4)	30.4 (26.1–34.7)	26.6 (22.9–30.3)	43.5 (39.0–48.0)	45.5 (40.6–50.4)	47.9 (42.4–53.4)	0.03
Sedentary behavior‖¶				14.8 (11.5–18.1)	9.8 (7.3–12.3)	9.3‡ (6.2–12.4)	24.6 (19.7–29.5)	20.9 (16.2–25.6)	18.3 (12.8–23.8)	0.69
Tobacco exposure#				19.0 (14.9–23.1)	9.7 (7.5–11.9)	6.5§ (4.7–8.3)	48.2 (41.5–54.9)	46.4 (41.3–51.5)	41.4 (35.5–47.3)	0.002
High sBP**	9.1 (5.4–12.0)	6.4 (4.0–9.4)	5.1 (2.2–8.0)	6.2 (4.2–8.2)	7.2 (5.2–9.2)	4.8 (3.0–6.6)	13.4 (10.1–16.7)	11.3 (7.8–14.8)	14.0 (9.7–18.3)	0.23
High non–HDL-C	11.8 (9.1–14.5)	12.0 (9.3–14.7)	12.2 (9.3–15.1)	12.0 (8.9–15.1)	11.8 (8.7–14.9)	13.6 (10.5–16.7)	26.2 (21.5–30.9)	23.0 (17.5–28.5)	26.7 (21.6–31.8)	0.91
Elevated HbA_{1c}‖				3.9 (2.7–5.1)	3.3 (2.1–4.5)	2.9 (1.5–4.3)	3.1 (1.5–4.7)	2.2 (1.0–3.4)	2.4 (0.6–4.2)	0.89
Elevated hs-CRP	2.6 (1.0–4.2)	3.7 (1.3–6.1)	2.5 (0.5–4.5)	3.2 (1.4–5.0)	2.4 (1.0–3.8)	2.3 (1.1–3.5)	5.0 (2.5–7.5)	4.6 (2.2–7.0)	3.3 (1.3–5.3)	0.91
Females										
Obesity	18.7 (16.0–21.4)	15.1 (12.0–18.2)	14.1 (10.2–18.0)	17.9 (14.0–21.8)	18.4 (15.3–21.5)	13.1‡ (10.4–15.8)	25.6 (20.9–30.3)	26.4 (21.1–31.7)	19.7 (12.8–26.6)	0.53
Central obesity	32.1 (28.0–36.2)	31.1 (26.0–36.2)	28.0 (22.5–33.5)	41.5 (36.6–46.4)	42.9 (38.2–47.6)	29.2§ (25.7–32.7)	56.1 (49.2–63.0)	56.9 (50.6–63.2)	47.8 (41.1–54.5)	0.22
Sedentary behavior‖¶				20.4 (16.9–23.9)	16.3 (13.0–19.6)	9.4§ (6.5–12.3)	27.0 (21.9–32.1)	21.4 (17.5–25.3)	19.1 (13.0–25.2)	0.31
Tobacco exposure#				14.1 (10.4–17.8)	10.4 (7.1–13.7)	5.9§ (3.9–7.9)	31.1 (24.8–37.4)	28.1 (22.4–33.8)	22.5 (17.2–27.8)	0.26
High sBP**	6.6 (5.4–12.0)	6.3 (4.0–9.4)	5.0 (2.2–8.0)	3.6 (2.2–5.0)	4.9 (2.5–7.3)	4.7 (3.1–6.3)	2.0 (0.6–3.4)	2.5 (0.9–4.1)	1.9 (0.1–3.7)	0.65
High non–HDL-C	16.9 (12.8–21.0)	12.2 (9.1–15.3)	13.9 (8.4–19.4)	12.6 (9.5–15.7)	11.5 (8.8–14.2)	8.7 (5.8–11.6)	23.4 (18.7–28.1)	24.7 (18.6–30.8)	15.8‡ (11.5–20.1)	0.31
Elevated HbA_{1c}‖				1.9 (0.9–2.9)	2.2 (1.2–3.2)	2.3 (0.7–3.9)	2.6 (1.4–3.8)	1.9 (0.3–3.5)	2.7 (0.5–4.9)	0.77
Elevated hs-CRP	3.5 (1.9–5.1)	3.3 (1.1–5.5)	0.6§ (−0.2 to 1.4)	4.2 (2.6–5.8)	4.0 (1.5–6.5)	1.7‡ (0.5–2.9)	9.0 (6.1–11.9)	7.8 (5.1–10.5)	8.3 (4.0–12.6)	0.13

Logistic regression models of CVD risk factors controlling for PIR, race/ethnicity, age, and PIR × age interaction. Values are calculated as predicted percentages (95% CI). T, tertile. †*P* value for PIR × age interaction calculated from *F* statistic based on design-corrected Wald χ^2 test. ‡$P < 0.05$ for difference between T1 and T3. §$P < 0.01$ for difference between T1 and T3. ‖Assessed for individuals aged ≥12 years. ¶Because of changes in physical activity assessment in 2007, analyses were restricted to data from NHANES 1999–2006. #Cases of cotinine >13 ng/mL among 6- to 11-year-olds too low to permit analyses. **Measured in individuals aged ≥8 years.



	T1	T2	T3	T1	T2	T3	T1	T2	T3
≥3 Risk Factors	5.0	1.5	0.7	2.5	2.3	2.0	14.7	13.1	14.5
2 Risk Factors	10.6	9.2	7.8	13.9	12.3	8.8	25.6	26.8	26.8
1 Risk Factor	33.8	31.3	19.4	36.6	30.3	31.0	38.0	36.3	32.8

	T1	T2	T3	T1	T2	T3	T1	T2	T3
≥3 Risk Factors	3.1	1.0	0.7	2.6	2.5	1.0	11.7	9.0	6.8
2 Risk Factors	13.8	10.7	7.5	14.7	13.5	8.1	23.6	23.0	20.7
1 Risk Factor	29.6	33.5	36.9	39.2	39.8	29.3	37.6	42.5	34.8

Figure 1—*Predicted prevalence (%)† of multiple risk factors for U.S. male* (A) *and female* (B) *NHWs, NHBs, and MAs aged 8–24 years‡ by PIR tertile (T) and age-group.* *P $<$ *0.05 for difference between T1 and T3 in having at least three risk factors (out of: central obesity, systolic hypertension, elevated hs-CRP, high non–HDL-C, and tobacco exposure).* †*Multinomial logistic regression models of multiple risk factors controlling for PIR, race/ethnicity, age, and PIR* $\times$ *age interaction. Values are calculated as predicted percentages. Interaction of PIR and age was nonsignificant for males* (P = *0.17) and females* (P = *0.49).* ‡*Age limited to* $\geq$*8 years because sBP was not measured in 6- to 7-year-olds.*

households below poverty were consistent with those of the U.S. Census Bureau (19). Also, although PIR is a robust indicator of current resources, we recognize that this national-level threshold cannot account for regional cost-of-living differences and does not comprehensively represent the multiple domains of SEP, each of which provides a unique lens on how disadvantage affects health. The limited number of per-survey observations precluded examination of CVD risk profile trends over time. Lastly, we focused on overarching SEP-CVD risk relationships and could not account for individual variability in growth, maternal and paternal BMI, and intrauterine exposures.

This study's strengths are the following: large, nationally representative, high-response rate sample; use of objective outcome measures; exposure (PIR) that integrates variations in household need and purchasing power over time; design-adjusted statistical methods; and stratification by age and sex to account for pubertal changes and/or socially patterned differences between sexes.

Implications

These findings showing greater preponderance of behavioral CVD risks among youths from lowest-income households provide two important inferences. First, viewed in the context of established trends, these data forewarn of recurring health and economic burdens. Second, this study offers insights into meaningful avenues for intervention, reinforcing that poor SEP hinders achievement of positive lifestyle choices. These implications are discussed separately.

The U.S. Census Bureau (19) estimates that during the past decade, 16–19% of those $<$18 years lived below the federal poverty level (for a family of four, annual household income $<$\$17,463 [year 2000] and $<$\$21,834 [year 2008]). This estimate has not varied greatly (range 15–23%) during the past 4 decades (10). Since the previous national-level reported data (8) collected 2 decades ago, our findings show persistent socioeconomic and health disparities among U.S. youth.

Early life socioeconomic conditions indirectly affect life course (adult SEP and health risks) (24) plus foretell low SEP over ensuing generations. Early life opportunities (e.g., education) shape future occupation, income, parity, personal (self-esteem and resilience) and social capital (relationships and networks), and investments in opportunities for the next generation (1). Moreover, metabolic, vascular, and orthopedic consequences associated with CVD risk cumulatively translate into high health care costs and potentially less productive life years, further reducing prospects of escaping low SEP.

Health-related behaviors are established early, and CVD risks persist into adulthood (25). Sedentary behaviors and tobacco exposure were common among adolescents but ominously twofold higher among young adults, suggesting that earlier intervention may circumvent future burdens. Also, national youth surveillance shows that previous declines in tobacco use are leveling off and positive weight-related behaviors have both been declining, emphasizing the need to reenergize prevention and promotion programs. However, the complexity of constraints faced by disadvantaged groups necessitate that CVD prevention policies address multiple levels (8,23). For example, since poverty contributes to low awareness of health risks, food insecurity, and restricted choice (21), regulations targeting price and content of foods and beverages are less meaningful without greater affordability of healthy alternatives. In a similar manner, neighborhood planning and safety are requisites for promoting physical activity. Lastly, to achieve population-level benefits, health promotion programs must reach low-income minority populations.

This study reports the most recent youth CVD risk factor distributions across SEP strata and isolates relationships between income disparities and health behaviors. These results are hypothesis generating—to reduce health and sociodemographic disparities nationally, policies that attenuate the effects of socioeconomic stressors must be judiciously tested to evaluate if they enhance the effectiveness of health promotion strategies. Finally, since our data predate the current economic

downturn that may impose greater disparities, it will be imperative to continue surveillance of disparities among children and youth.

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M.K.A. and K.M.B. designed the study, conducted all analyses, interpreted data, prepared and reviewed the manuscript, had full access to all data in the study, and take full responsibility for the integrity of data and the accuracy of data analysis. G.L.B. interpreted data; provided guidance for sophisticated analyses; and wrote, reviewed, and edited the manuscript. M.R.S. interpreted data, and prepared and reviewed the manuscript. L.B. and K.M.V.N. interpreted data; provided guidance for sophisticated analyses; and wrote, reviewed, and edited the manuscript. G.I. designed the study, conducted all analyses, interpreted data, prepared and reviewed the manuscript, had full access to all data in the study, and takes full responsibility for the integrity of data and the accuracy of data analysis.

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